LETTERS TO THE EDITOR

https://doi.org/10.1093/europace/euac109
Published online 19 July 2022

Low voltage with high suspicion in athlete's heart

We have read with great interest the manuscript recently published in your journal by Zorzi *et al.*[1] about the prevalence and significance of the presence of isolated low voltages in the electrocardiogram (ECG) of young athletes. About this work, several aspects are of interest, especially the proposal to include this electrocardiographic finding among the infrequent but potentially pathological changes in the athlete's heart. Based on our experience with similar two triathletes with low voltage, a 40-year-old male with syncope and ventricular tachycardia and a 29-year-old female with exertional premature ventricular contractions both with subepicardial inferolateral late gadolinium enhancement (LGE) and negative genetic test that ruled out left ventricle arrhythmogenic cardiomyopathy—according to the Padua criteria—we raised several questions related to the published manuscript.[2]

In this, not all athletes with low voltage have magnetic resonance imaging (MRI) performed but only those with ventricular tachycardias, it remains in the air therefore if the rest would also have LGE and if this could be the substrate of a low electrocardiographic voltage in the ECGs. On the other hand, once the diagnosis of left-sided dysplasia has been ruled out, it is worth asking whether the LGE present in our patients, in both at the same location and with a similar electrocardiographic expression, could be the result of an excessive training responsible of a non-ischaemic myocardial damage in over-trained susceptible individuals or just the result of a previous myocarditis. This takes us even further: would it be necessary to perform MRI on all the athletes with low voltage or at least a closer periodic follow-up that includes Holter monitoring and exercise stress test? Are we facing a new expression of the heart of a high-performance athlete? The real significance of LGE described in athletes is unclear, as mentioned by Domenech-Ximenos *et al.*,[3] the presence of LGE indicates that the local matrix and fibre structure has changed, similar to what happens in histologic fibrosis and scar, otherwise it has been shown that acute profound exercise-related oxidative stress and inflammation may impair endothelial function and arterial stiffness and this phenomena could be responsible for fibrosis.[4,5]

We propose the creation of national and international case registries that would undoubtedly improve our knowledge of the athlete's heart and its adaptive and non-benign-adaptative changes. The presence of low voltage, according to the authors, may be a not-so-benign finding requiring a more depth study and a closer periodical follow-up.

Conflict of interest: None declared.

References

1. Zorzi A, Bettella N, Tatangelo M, Del Monte A, Vessella T, Poscolieri B, *et al.* Prevalence and clinical significance of isolated low QRS voltages in young athletes. *Europace* 2022;**24**:1484–95.
2. Corrado D, Perazzolo Marra M, Zorzi A, Beffagna G, Cipriani A, De Lazzari M, *et al.* Diagnosis of arrhythmogenic cardiomyopathy: the Padua criteria. *Int J Cardiol* 2020;**319**:106–14.
3. Domenech-Ximenos B, Sanz-de la Garza M, Prat-González S, Sepúlveda-Martínez A, Crispi F, Duran-Fernandez K, *et al.* Prevalence and pattern of cardiovascular magnetic resonance late gadolinium enhancement in highly trained endurance athletes. *J Cardiovasc Magn Reson* 2020;**22**:62.
4. Scherr J, Braun S, Schuster T, Hartmann C, Moehlenkamp S, Wolfarth B, *et al.* 72-h Kinetics of high-sensitive troponin T and inflammatory markers after marathon. *Med Sci Sports Exerc* 2011;**43**:1819–27.
5. La Gerche A. The potential cardiotoxic effects of exercise. *Can J Cardiol* 2016;**32**:421–8.

Antonio Adeba[1], María Martín[1,2]*, and José Julián Rodríguez Reguero[1,2]
[1]Area de Gestión Clínica del Corazón, Hospital Universitario Central de Asturias Oviedo, Oviedo, Spain; and [2]Instituto de Investigación Biosanitaria del Principado de Asturias (ISPA), Oviedo, Spain
*Corresponding author. *E-mail address*: mmartinf7@hotmail.com

https://doi.org/10.1093/europace/euac110
Published online 19 July 2022

Low voltage with high suspicion in athlete's heart: Authors' reply

We thank Adeba *et al.*[1] for their interest in our study. Based on their experience with two athletes with low QRS voltages (LQRSVs), complex ventricular arrhythmias, and underlying non-ischaemic left ventricular scars (NILVSs), the authors of the letter to the editor raised two important questions: (i) should we perform cardiac magnetic resonance (CMR) to all athletes with LQRSV regardless of associated clinical abnormalities and (ii) can NILVS be the result of exercise-induced cardiac damage? We still do not have an answer to the second question and we agree that more data need to be collected. However, we would like to address the first point.

The background of our study[2] was that the 2017 International recommendations for interpretation of the athlete's electrocardiogram (ECG) did not mention LQRSV,[3] although this sign can be associated with potentially at-risk myocardial substrates (particularly the NILVs).[4] The primary study result was that LQRSVs are uncommon in healthy athletes at difference to patients with cardiomyopathies; the secondary was that in a proportion of athletes, LQRSV were associated with pathological myocardial substrates on CMR. These findings lead to classify this previously unaddressed ECG pattern among the uncommon ECG abnormalities requiring further tests. The problem is what tests.

Our observational study reflecting the current clinical practice was not designed to assess the prevalence of underlying pathological myocardial substrates in the entire population of athletes with LQRSV, but only in those with associated clinical abnormalities at second-line investigations (family history, resting and exercise ECG, 24 h ambulatory ECG monitoring, and echocardiography) prompting CMR prescription. For this reason, we cannot exclude that LQRSV may be occasionally the only clinical manifestation of an at-risk cardiac disease and that a CMR study might be reasonable in athletes with isolated LQRSV. However, this recommendation cannot be drawn based on the findings of our[2] and a previous study.[5]

Based on the available data, we propose that athletes with LQRSVs should be first investigated by additional echocardiography, exercise testing, and 24 h ECG Holter monitoring, and those with abnormalities found by these tests undergo CMR for exclusion of a cardiomyopathy. This strategy is a first but important step towards the best management (in terms of both efficacy and cost-effectiveness) of an athlete with isolated LQRSV, if one considers that up to now this ECG pattern was overlooked. The present proposal to reserve a further CMR scan for those athletes with associated abnormalities, besides the still limited data, is also motivated by the awareness that systematic CMR in all athletes with LQRSV (despite its low prevalence in the athletic

population) may be hardly sustainable in the setting of a mass screening because of logistics, feasibility, and costs. Further studies would be needed to assess whether indications to CMR in athletes with LQRSV should be expanded.

Conflict of interest: None declared.

References

1. Adeba A, Martìn M, Rodrìguez Reguero JJ. Low voltage with high suspicion in athlete's heart. *Europace* 2022;**24**:1540.
2. Zorzi A, Bettella N, Tatangelo M, Del Monte A, Vessella T, Poscolieri B, *et al.* Prevalence and clinical significance of isolated low QRS voltages in young athletes. *Europace* 2022;**24**:1484–95.
3. Sharma S, Drezner JA, Baggish A, Papadakis M, Wilson MG, Prutkin JM, *et al.* International recommendations for electrocardiographic interpretation in athletes. *Eur Heart J* 2018;**39**:1466–80.
4. De Lazzari M, Zorzi A, Cipriani A, Susana A, Mastella G, Rizzo A, *et al.* Relationship between electrocardiographic findings and cardiac magnetic resonance phenotypes in arrhythmogenic cardiomyopathy. *J Am Heart Assoc* 2018;**7**:e009855.
5. Mango F, Caselli S, Luchetti A, Pelliccia A. Low QRS voltages in Olympic athletes: prevalence and clinical correlates. *Eur J Prev Cardiol* 2020;**27**:1542–8.

Alessandro Zorzi and Domenico Corrado*

Department of Cardiac, Thoracic and Vascular Sciences, University of Padova, Via Giustiniani, 2, 35121 Padova, Italy

*Corresponding author. Tel: +39 0 49821245; fax: +39 0 498212309. *E-mail address:* domenico.corrado@unipd.it

https://doi.org/10.1093/europace/euac074
Published online 31 May 2022

The importance of individualized multimodality imaging-guided methods for selected patients in cardiac resynchronization therapy

We read the article by Fyenbo *et al.*[1] titled 'Long-term outcomes in a randomized controlled trial of multimodality imaging-guided left ventricular lead placement in cardiac resynchronization therapy (CRT)' with great interest. In this very well-designed study, it was reported that individualized multimodality imaging-guided left ventricular (LV) lead placement does not reduce the risk of heart failure (HF)-associated hospitalization or all-cause death after a median 6.7 years of follow-up. We believe that it would be useful to determine and classify which patients should undergo individualized multimodality imaging before CRT implantation. A recent study reported that only a combination of the absence of scars (detected using late gadolinium-fortified cardiac magnetic resonance) and response to CRT is associated with a positive long-term survival.[2] About one-third of CRT recipients are 'non-responders'.[3] Varma *et al.*[3] reported that the current health expenditures associated with CRT non-responder management were one of the highest among any groups of patients with HF. Individualized multimodality imaging-guided strategies can also be used in cases of suspected coronary venous diseases that are less known and actively investigated and may cause CRT non-response. In an autopsy-based study on cadavers, Watanabe *et al*. observed two types of coronary muscular bridges: a bridged artery type and a bridged artery and vein type. In the second type, both the coronary artery and vein were covered with myocardium.[4] Considering that muscular bridges most commonly develop in the left coronary arterial system, especially in the left anterior descending artery,[5] it should be kept in mind that coronary venous muscular bridges may also be found before CRT implantation. When placing the LV lead, if the coronary venous stenosis is connected to the muscular coronary venous bridge, the coronary venous angioplasty will not be effective and may cause CRT non-response. Furthermore, stent implantation is not recommended for the treatment of muscular bridges as the risk of thrombosis, restenosis, and perforation is high.[5] Imaging examinations before CRT implantation and imaging guidance during implantation are the only diagnostic tools that can help us identify lesser-known coronary venous diseases.

Individualized multimodality imaging-guided strategies in selected patient groups planned for CRT implantation may reduce CRT non-response, health-related expenditures and allow clinicians to take a close look at coronary venous diseases.

Conflict of interest: None declared.

References

1. Fyenbo DB, Sommer A, Nørgaard BL, Kronborg MB, Kristensen J, Gerdes C, *et al.* Long-term outcomes in a randomized controlled trial of multimodality imaging-guided left ventricular lead placement in cardiac resynchronization therapy. *Europace* 2022;**24**:828–834.
2. Linhart M, Doltra A, Acosta J, Borràs R, Jáuregui B, Fernández-Armenta J, *et al.* Ventricular arrhythmia risk is associated with myocardial scar but not with response to cardiac resynchronization therapy. *Europace* 2020;**22**:1391–400.
3. Varma N, Auricchio A, Connolly AT, Boehmer J, Bahu M, Costanzo MR, *et al.* The cost of non-response to cardiac resynchronization therapy: characterizing heart failure events following cardiac resynchronization therapy. *Europace* 2021;**23**:1586–1595.
4. Watanabe Y, Arakawa T, Kageyama I, Aizawa Y, Kumaki K, Miki A, *et al.* Gross anatomical study on the human myocardial bridges with special reference to the spatial relationship among coronary arteries, cardiac veins, and autonomic nerves. *Clin Anat* 2016;**29**:333–341.
5. Roberts W, Charles SM, Ang C, Holda MK, Walocha J, Lachman N, *et al.* Myocardial bridges: a meta-analysis. *Clin Anat* 2021;**34**:685–709.

Ercan Akşit[1]*, Uğur Küçük[1], Servet Altay[2], Gökay Taylan[2], and Halil Fatih Aşgün[3]

[1]Canakkale Onsekiz Mart University Faculty of Medicine, Department of Cardiology, Barbaros Street Terzioglu Campus B Block No: 4, Canakkale, Turkey; [2]Trakya University Faculty of Medicine, Department of Cardiology, Edirne, Turkey; and [3]Canakkale Onsekiz Mart University Faculty of Medicine, Department of Cardiovascular Surgery, Canakkale, Turkey

*Corresponding author: Tel: +90 286 263 59 50, fax: +90 286 263 59 56. *E-mail address:* ercanaksit@comu.edu.tr

https://doi.org/10.1093/europace/euac075
Published online 14 June 2022

The importance of individualized multimodality imaging-guided methods for selected patients in cardiac resynchronization therapy: Authors' reply

We appreciate the comments made by our colleagues regarding our recently published paper, 'Long-term outcomes in a randomized controlled trial of multimodality imaging-guided left ventricular (LV) lead placement in cardiac resynchronization therapy'[1] and the opportunity to respond to their comments. We reported that multimodality imaging-guided LV lead placement towards the coronary sinus branch closest to the latest mechanically activated non-scarred myocardial segment did not reduce the risk of heart failure hospitalization or all-cause death as compared with routine LV lead placement after long-term follow-up of median 6.7 years in patients treated with cardiac resynchronization therapy (CRT).[1] It is well recognized that a significant proportion of CRT recipients do not respond with clinical improvement and that health expenditures associated with CRT non-responder management are considerable.[2] The commentators therefore suggested that it would be useful to determine and classify which patients should undergo individualized multimodality imaging before CRT implantation.[3] Specifically, the role of myocardial